Sudden death in turkeys with perirenal hemorrhage: Field and laboratory findings

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Abstract

Sudden death associated with perirenal hemorrhage, pulmonary congestion and edema, splenic enlargement and mottling, and hepatic congestion affected rapidly-growing, apparently healthy male turkeys. Concurrent lesions of other diseases occurred in some birds. No causative agent was identified by bacteriological examination.

The number of diagnoses of this condition has been increasing in recent years. Mortality usually occurs in males at 8–13 weeks of age. This condition was seen throughout Alberta and was not restricted to poults from any specific hatchery, or on any specific feed. Fast weight gain, continuous lighting programs, crowding, and hyperactivity are possible important etiological factors. Various treatments have been used with no significant effect on mortality from the condition.

Résumé

Mort soudaine avec hémorragie périrénale chez des dindons : renseignements cliniques sur le terrain et résultats de laboratoire

Mort soudaine caractérisée par une hémorragie périrénale, de la congestion et de l'œdème pulmonaire, de la congestion hépatique et une rate volumineuse d'apparence marbrée fut observée chez des dindons mâles, apparemment en santé, en phase de croissance rapide. Certains oiseaux présentèrent aussi des lésions de diverses maladies. Aucun agent bactérien n'a été identifié. Le nombre de cas diagnostiqués de cette maladie a augmenté depuis ces dernières années. La mort survient en général chez les mâles âgés de 8 à 13 semaines. Cette condition se rencontre partout en Alberta et n'est pas restreinte à des oiseaux de certains couvoirs ou reliée à une nourriture spécifique.

Le gain rapide de poids, les programmes d'éclairage continu, l'entassement et l'hyperactivité sont considérés comme facteurs étiologiques possibles. Différents traitements ont été administrés sans effet significatif sur le taux de mortalité.

(Traduit par Dr Thérèse Lanthier)

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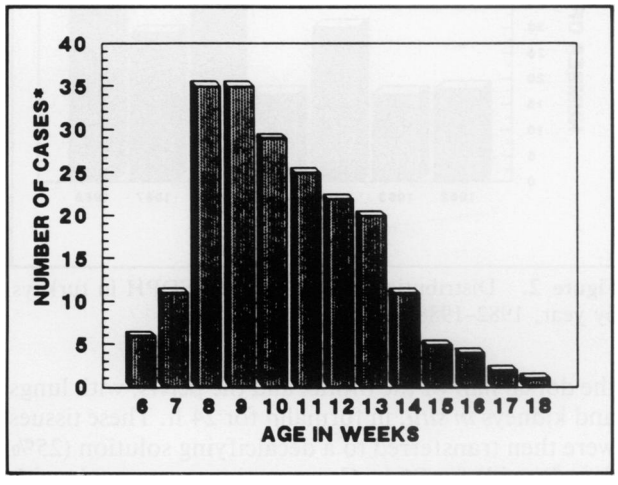

Figure 1. Age of turkeys with sudden death. *(Age was not mentioned in 17 cases.)

Introduction

Sudden death with perirenal hemorrhage (SDPH) causes significant economic loss to commercial turkey operations in Alberta and is widely diagnosed in Canada (1). A search of the literature failed to reveal any detailed study on this condition. The terms sudden death in turkeys (2), hypertensive angiopathy (2–4), and sporadic renal hemorrhage (5) have been used to describe similar if not identical syndromes in turkeys. This confusing terminology has occurred because the etiology and pathogenesis of the condition are unknown. An angiopathy has been described in the small arteries in the spleen and other tissues of affected birds (4). Suggested causes for the angiopathy have included hypertension (2) and bacterial (3) or viral infections (4). We describe herein SDPH in turkeys in Alberta from 1982–1988.

Materials and methods

Diagnostic records of the Animal Health Laboratory, Edmonton, between 1982 and 1988 were reviewed. Detailed histological and bacteriological studies were conducted on 21 turkeys in 1988.

Histology

Samples from 10 turkeys with mild to severe lesions of SDPH were collected approximately 4–6 h after death (judged from case history and appearance of carcass). Samples were also collected from two apparently healthy turkeys for comparison. These two turkeys were obtained live and were killed by breaking their neck with a burdizzo immediately prior to necropsy. From each bird, pieces of spleen, adrenal glands, bursa of Fabricius, thymus, heart, and liver were fixed in 10% neutral buffered formalin. The lungs and kidneys were fixed intact by immersion of

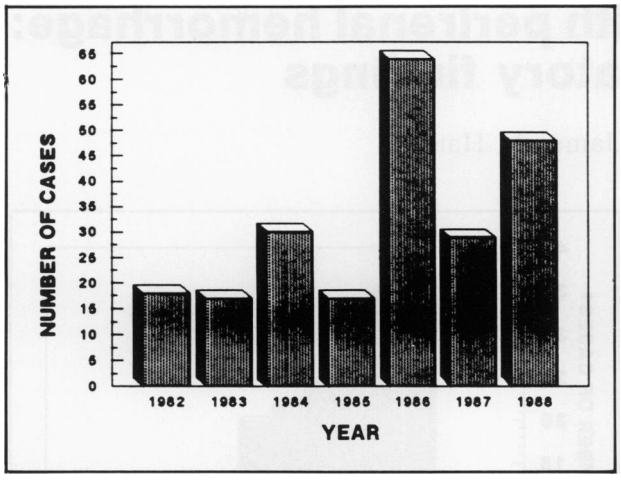

Figure 2. Distribution of 223 cases of SDPH in turkeys by year, 1982–1988.

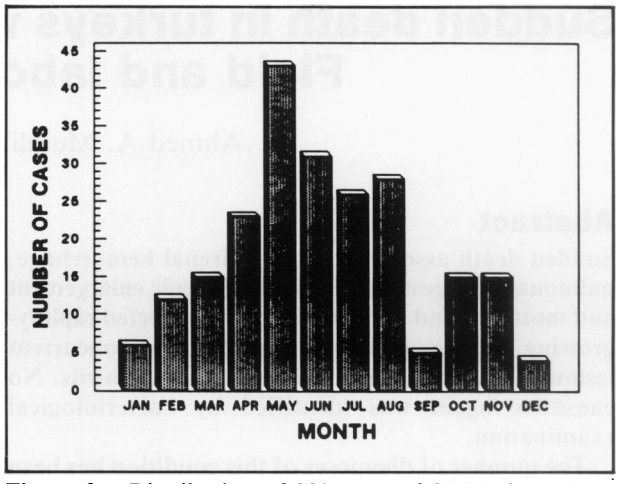

Figure 3. Distribution of 223 cases of SDPH in turkeys by month, 1982–1988.

the dorsal half of the thorax and the pelvis, with lungs and kidneys *in situ*, in formalin for 24 h. These tissues were then transferred to a decalcifying solution (25% formic acid) for 96 h. Cross sections were made with a sharp knife through the entire thickness of the remaining thorax and pelvis. Six transverse sections were taken from each lung. Both lungs were sampled in a similar fashion to obtain tissue from identical sites. One transverse section was taken from each renal lobe bilaterally. Care was taken while sampling to keep the renal surface membrane intact and a rim of the surrounding bone was included in the section. All tissue samples were processed, sectioned, and stained with hematoxylin and eosin by routine techniques.

Bacteriology

Intestine, liver, and heart blood from 11 turkeys with lesions of SDPH, different from those used for histology, were cultured for aerobic and anaerobic bacteria. Cultures were made on 5% sheep blood agar plates. The aerobic cultures were incubated at 35–37°C for 24 h while the anaerobic cultures were incubated at 35–37°C under 10% CO_2 for 48 h.

Questionnaire and farm visits

A questionnaire was sent to all major commercial turkey producers in Alberta (Table 1). Information to supplement the questionnaire was obtained on farm visits and from some feed companies.

Results

Between 1982 and 1988 SDPH was diagnosed in 208 submissions (or cases, as referred to in Figures 1–3) from commercial farms, 13 submissions from research flocks, and two submissions from backyard flocks for a total of 468 turkeys; 413 of these turkeys had lesions only of SDPH. The remaining 55 turkeys had concurrent lesions of other diseases including granulomatous pneumonia due to aspergillosis or aspirated food (16), aortic rupture (14), candidiasis (14), colibacillosis (6), and hemorrhagic enteritis (5). Sudden death occurred more frequently in male turkeys (221 submissions) than in females (two submissions). Mortality due to SDPH occurred most commonly at 8–13 weeks of age (Figure 1). Total mortality due to SDPH alone was estimated to reach 6% in some turkey flocks. This condition was diagnosed in each year and the number of diagnoses each year has increased from 1982–1988 (Figure 2). The highest prevalence of SDPH occurred in April–August; however, it was diagnosed throughout the year (Figure 3). Affected flocks varied in size from 200 to 18,000 birds (Table 2).

Sudden death with perirenal hemorrhage affected rapidly growing, apparently healthy male turkeys between 6–18 weeks of age. Affected birds were in excellent body condition and frequently had partially filled crops and gizzards. The most characteristic lesion was mild to severe perirenal hemorrhage. In mild hemorrhage, only one or two renal lobes, usually the cranial lobes, were partially or totally covered with blood. In severe hemorrhage, the blood covered most of the renal lobes (Figure 4). The kidney appeared normal (Figure 5) and the accumulated blood was clotted and occupied the space between the renal surface membrane and the peritoneum with the exception of three birds; in these, rupture of the renal surface membrane had occurred, resulting in clotted blood in the abdominal cavity.

Pulmonary congestion and edema affecting the entire lung were seen consistently in turkeys recently dead from SDPH. These changes were more pronounced in turkeys dead for a few hours, and were more severe in the ventral parts of lungs. There was red fluid in the thoracic cavity after the lungs were removed (Figure 6). The liver was dark brown, enlarged, and firm in most birds examined. Blood oozed from cut hepatic surfaces, and blood vessels were congested. In most birds examined, the spleen was enlarged and mottled with prominent, 1 mm white foci and 3–5 mm diameter red patches. In the rest of the birds, the spleen was of normal size but was mottled. The small intestines were distended with white, thick mucus; however, the intestinal mucosa appeared normal. The heart, bursa of Fabricius, thymus, adrenal glands, testes, and pancreas appeared slightly congested. Bone strength was satisfactory as assessed by manually breaking both tibias.

Table 1. Results of questionnaire on sudden death in turkeys with perirenal hemorrhage in Alberta

	Farms with SDPH problem	Farms without SDPH problem
Number of farms	30	9
Strain of turkeys		
— heavy	30	0
— broiler	0	9
Sex — male	30	6
— female	0	3
Age when SDPH started and duration		
— 8 wk-market	30	N/A
Hatchery source		
— A	8	7
— B	5	2
— C,D,E,F,G,H	1,1,8,3,2,2	0
Feed source		
— A	4	1
— B	11	5
— C	1	2
— D	1	0
— E,F,G,H,I,J,K	1,1,1,2,2,1,1	0
— home made	4	1
Form of feed		
— pelleted	22	7
— crumbles and mash	8	2
Age of turkeys when marketed	16–19 wk	14–16 wk
Weight of turkeys at market	10–14 kg	6.5–8.5 kg
First and last check of flock		
— morning 6–8am	30	9
— night 5–12pm	30	9
Approximate time of death		
— night	17	N/A
— day	5	
— anytime	3	
— not mentioned	5	
Age at which turkeys are moved between houses	5–7 wk	5–7 wk
Relation between SDPH and moving birds		
— yes	22	N/A
— not mentioned	8	
Relation between SDPH and change of feed		
— no	28	N/A
— not mentioned	2	
Relation between SDPH and other management changes		
— no	30	N/A
Type of brooding house		
— closed	30	9
Type of growing house and lighting		
— closed and fluorescent	2	0
— closed and incandescent	0	9
— semi-closed and incandescent	28	0
Lights		
— on at night	16	0
Litter used		
— straw	15	5
— shavings	14	2
— paper	1	2
Water source		
— well	17	6
— dugout	6	3
— not mentioned	7	0
Vaccine history		
— no vaccination	24	8
— cholera	2	0
— hemorrhagic enteritis	4	1
Treatment		
— farmers tried medication	20	N/A
— did not medicate	4	
— not mentioned	6	

Figure 4. Kidneys and testes *in situ* of a 10-week-old turkey with SDPH. Note hemorrhage covering most of the renal lobes (arrows).

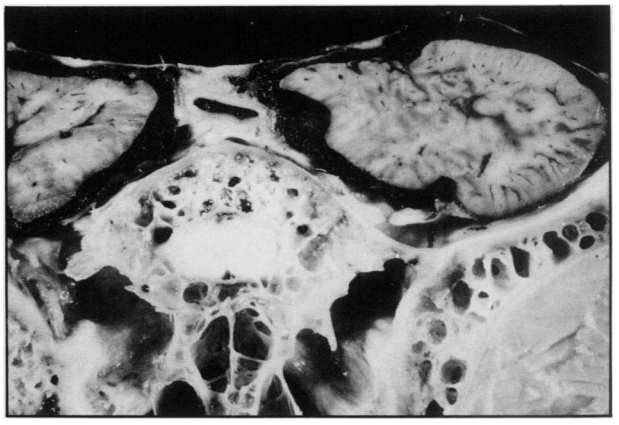

Figure 5. Cross section of a formalin-fixed and decalcified pelvic region from a 10-week-old turkey with SDPH. Note accumulation of blood beneath the renal surface membrane. The kidney itself is apparently normal.

Histology

Histological findings confirmed the gross findings. No inflammatory cells were noted. Endothelial cells of blood vessels from all tissues protruded into the lumen and presented a toothed appearance. The tunica media in the same vessels was disoriented with increased empty spaces between fibers. These vessel wall changes were seen in birds with lesions of SDPH as well as in apparently healthy turkeys.

Bacteriology

No significant organism was isolated from the liver, intestine, or heart blood of any bird with the exception of a scant growth of *Clostridium perfringens* from the intestine of three birds.

Questionnaire and farm visits

Of 54 questionnaires submitted to all major commercial turkey producers in Alberta, 39 replies were received. Thirty of these 39 producers replied that SDPH had been diagnosed on their farms. Laboratory diagnostic records confirmed these data.

Results of the questionnaire are summarized in Table 1. Sudden death affected primarily heavy strain, male turkeys. Five farmers reported having seen birds at the time of death. These affected birds showed disoriented activity and wing beating immediately before they died. One farmer observed general hyperactivity of the flock, with birds being more noisy than usual. The five farmers mentioned above also reported having opened birds that died of SDPH and observed blood around the kidneys. The majority of farmers reported mortality from SDPH within two to three weeks of moving turkeys from brooding to growing houses. Mortality from SDPH was also noted if birds were fighting, and was increased during hot and humid weather. Mortality from SDPH occurred mostly at night. One farmer reported that mortality from SDPH coincided with sunrise; he confirmed his observation by checking the flock before and after sunrise. Another observed that turkeys dead from SDPH were found in the brightest spots in the barn.

Farms with SDPH problems marketed their turkeys at a later age and heavier weight than farms without SDPH problems.

In all farms (with or without SDPH problems) poults were raised in total confinement during the brooding period from day 1 until five to seven weeks of age and were then moved to the growing houses. The growing houses in 28/30 farms with SDPH problems were semi-closed (birds having access to the outside), utilizing daylight and incandescent light at night (3.3 watt/m^2). Two barns with SDPH problems (2/30) were totally closed; however, poults were raised under continuous fluorescent light (3.3 watt/m^2) from day 1 to market age. Flocks with SDPH problems tended to be more crowded than flocks without SDPH problems.

Farmers who did not report SDPH as a problem raised turkeys in total confinement from day 1 until marketing and used a different lighting regimen. During the first three days of life, bright lighting (2.2 watt/m^2) was used, then lights were dimmed to a minimum (0.5 watt/m^2) for the next six weeks. Light intensity was then gradually increased over several weeks to reach a maximum of 2.2 watt/m^2 near market age at 14–16 weeks.

Factors such as litter type, water source, hatchery and feed source, vaccines administered, and change of feed were seen with relatively equal frequency on farms with and without SDPH problems.

Information obtained from four major Alberta-based feed companies with regard to protein content, energy level, and recommended feeding schedule is given in Table 3. The ratio of different grains in the diet varied depending on their availability and cost. Sodium chloride content in the feed varied between 0.5 and 1%. Pelleted feed was by far the most commonly used, with crumbles and mash feed used occasionally during certain weeks of the growing period on a few farms. Five farmers noted that high energy of commercial feed seemed to accentuate mortality from SDPH. One of these farmers reported to have reduced mortality from SDPH by feeding a low-energy, home-made diet.

Table 2. Flock size and prevalence of SDPH[a]

Flock size	Number of submissions	Number of flocks	Flock size	Number of submissions	Number of flocks
200–999	6	4	6000–6999	24	14
1000–1999	10	7	7000–7999	23	14
2000–2999	23	14	8000–8999	7	3
3000–3999	28	14	9000–9999	17	7
4000–4999	13	5	10000–10999	2	2
5000–5999	11	9	11000–18000	7	3

[a]Flock size was not mentioned in 52 submissions

The feed used on farms with or without SDPH problems contained either amprolium or dibutylin dilaurate as a coccidiostat and nifursol as a histostat. In some cases birds were on antimicrobial drugs such as neomycin/tetracycline, furazolidone, or sulfonamides at the time of mortality. Mortality from SDPH continued until marketing; feed was free of any medication for the last two weeks.

Various treatment regimens including calcium, aspirin, ascorbic acid, copper sulfate, vitamin-mineral mix, and different antibiotics such as tetracycline, bacitracin, and neomycin were reported to have no significant effect in reducing mortality from SDPH.

Discussion

The most typical lesion in turkeys with SDPH is perirenal hemorrhage. It is not clear why hemorrhage is associated with the kidneys, but the source of the hemorrhage appears to be vessel rupture (5). Death of affected turkeys is apparently not due to hemorrhage alone, based on the small volume of lost blood. This loss may even occur as a terminal event and, possibly, pulmonary congestion and edema play a more significant role as a cause of death of affected birds. These events may cause increased concentrations of carbon dioxide in the blood, leading to respiratory acidosis and secondary hyperkalemia, with resultant ventricular fibrillation and death.

Fast weight gain seems to be one of the possible factors increasing susceptibility to SDPH. Sudden death affected primarily male heavy turkeys; these strains are known for their fast growth rate and large body size. Birds dead from SDPH were well fleshed and some farmers regarded SDPH as an indication of good flock health and fast growth rate as in the case of sudden death syndrome (flip-over) in broiler chickens. Farms with SDPH problems marketed their turkeys at 10–14 kg (age 16–19 weeks). Sudden death was not a problem in farms marketing turkeys at 6.5–8.5 kg (age 14–16 weeks). Although the market age in these two groups of farms is slightly different, the weight variation between them is great.

Concurrent disease conditions such as pneumonia and colibacillosis as noted in some birds in the present study may have further impaired tissue oxygenation and increased mortality from SDPH. Hot and humid weather, and excitement created by birds fighting and moving, create a state of "stress" and may result in an increased demand for oxygen and subsequent heart failure. Similarly, crowding tends to increase "social stress". Continuous lighting programs may promote increased feed intake and therefore increased growth rate.

Figure 6. Pulmonary congestion and edema in a 10-week-old turkey with SDPH. Note fluid in the thoracic cavity (arrows).

Farmers who reported no SDPH problem raised female or broiler turkeys and employed different husbandry practices. Their barns were totally closed, and they adopted a well-controlled "step-up" lighting schedule.

Another possible cause of death is endotoxic shock. Birds generally had full gastrointestinal tracts. It is possible that fermentation and liberation of a toxin occurred in the digestive tract. It has been speculated that the cause of SDPH may be infection with *Clostridium perfringens* (3).

The possibility that SDPH is an early manifestation of aortic rupture is doubtful. Based on our laboratory records and field observations, the incidence of aortic rupture in turkeys has greatly declined in recent years in Alberta. We assume that achieving the desired market weight (around 12 kg) at an early age (around 18 weeks) before sexual maturity is an important reason for this decrease. For this reason, hypertension may not have a role in the pathogenesis of SDPH.

Angiopathy characterized by endothelial hyperplasia, necrosis of the media, and hyperplasia of smooth muscle was associated with various clinicopathological conditions in turkeys (6) and was later associated with SDPH as well (2). Such vascular changes were not seen in turkeys in the present study. Vascular wall changes observed in this study are considered to have been artifacts resulting from agonal vascular contraction possibly by the action of the fixa-

Table 3. Protein and energy levels in turkey feed from different feed companies (1–4) in Alberta, and feeding schedule recommended by the company

1	2	3	4	HM
30 p 3030 e 0–4 wk	29 p 2780 e 0–3 wk	28 p 2800 e 0–3 wk	28 p 2800 e 0–2.5 wk	29–31 p NA 0–3 wk
28 p 3095 e 4–7 wk	25 p 2880 e 4–7.5 wk	26 p 2900 e 3–7 wk	25 p 2900 e 2.5–5.5 wk	25–29 p NA 3–6 wk
24 p 3165 e 7–9 wk	22 p 3000 e 7.5–10 wk	23 p 3000 e 7–11 wk	23 p 2950 e 5.5–7.5 wk	21–23 p NA 6–14 wk
21 p 3235 e 9–11 wk	19 p 3000 e 11–13 wk	20 p 3000 e 11–14 wk	20 p 2950 e 7.5–11.5 wk	14–16 NA 14-market
19 p 3270 e 11–14 wk	17 p 3125 e 14–16 wk	17 p 3100 e 14–18 wk	17 p 2950 e 11.5–15 wk	
17 p 3300 e 14 to market	14 p 3125 e 17 to market	14 p 3200 e 18 to market	15 p 3060 e 15 to market	

p Protein percentage
e Energy (kilocalorie/kilogram)
HM Home made. Values represent highest and lowest levels provided by 4 producers
NA Not available
wk Age of turkeys in weeks

tive (7). Because of the questionable role of angiopathy in this condition and because of the lack of evidence of hypertension, it is more appropriate to call this condition "sudden death in turkeys with perirenal hemorrhage" since these are the characteristic features of this condition.

Our limited bacteriological examinations revealed no causative pathogen. However, other disease conditions may occur concurrently in birds with SDPH lesions as described above, and mortality would be expected to decrease upon use of antibiotics if a bacterial disease was involved. This may explain the successful response to antibiotics observed by other workers (3).

Sudden death in turkeys is considered of significant economic importance due to loss of many good birds in the flock with mortality due to SDPH reaching 6%. This condition is widely diagnosed in Alberta and is seen throughout the year. The high prevalence of SDPH in April–August may be explained by the fact that most producers start their flocks between early March and May. Between the end of April and early August, these turkeys are 8–13 weeks, the age at which most mortality from SDPH occurs. Moreover, during this period birds are moved out of the closed brooding houses to range houses with access to the outside. Hot and humid weather during summer months may further aggravate the problem. During the cold winter months, many farms stop raising turkeys. This may explain the low prevalence of SDPH during the September–February period. The distribution of cases by year and month is based on birds submitted by farmers to the laboratory for diagnosis and it may not reflect the exact situation in the field. Some farmers do postmortem examinations on their dead turkeys and make a diagnosis based on observations and knowledge learned from previous submissions.

In conclusion, it seems that, with the exception of perirenal hemorrhage, the clinical, histological, and bacteriological findings in turkeys with SDPH are similar to those seen in chickens with sudden death syndrome (flip-over). Furthermore, it seems that fast weight gain, which is a necessary economic strategy, may be a major factor in this problem. Decreasing the growth rate of these birds may be the only way to prevent the occurrence of SDPH at the present time.

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